Ruptured Intracranial Carotid Artery Aneurysm with Fatal Epistaxis

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Intracranial aneurysms are primarily a neurosurgical problem. The patient with an intracranial aneurysm may be seen initially, however, by the ophthalmologist or the otolaryngologist. It is the purpose of this paper to review the subject of traumatic internal carotid artery aneurysm, show how epistaxis may be a conspicuous part of the clinical features, and present an illustrative case.

Anatomy

In order to comprehend fully the symptoms produced by the intracranial aneurysm, a brief review of the intracranial course of the internal carotid artery is indicated.

As the internal carotid artery ascends into the carotid canal, it lies anteroinferior to the tympanum and cochlea, posteromedial to the Eustachian tube and the canal for the tensor tympani muscle (Fig. 1). Only a very thin lamella of bone separates the artery in its canal from the Eustachian tube and tympanum (Fig. 2) and this lamella frequently is found to be perforated. As the artery emerges from the carotid canal, it is found to be adjacent, just below, and medial to the trigeminal ganglion.

The artery runs forward, piercing the external layer of the dura mater in the lateral wall of the cavernous sinus. The oculomotor, trochlear, and abducens nerves and the ophthalmic division of the trigeminal nerve are found lateral to the artery within the sinus (Fig. 3).

As it approaches the lesser wing of the sphenoid bone, it turns superior to the medial side of the anterior clinoid process, pierces the inner layer of the dura mater and the arachnoid, and runs backward, fre-

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Fig. 1.—Oblique sagittal section through temporal bone.
quently forming a groove in the lateral wall of the sphenoid sinus (Figs. 3 and 4). It also comes in close relation with the inferior surface of the optic nerve just behind the optic foramen in this area. In its posterior course it comes between the optic and oculomotor nerves and finally turns upward at some distance from the optic chiasma to divide into the anterior and middle cerebral arteries. Here again, it is important to note the intimate relationship between the carotid artery and sphenoid sinus. Van Alyea\(^1\) emphasizes that “irregularities in the wall of some sinuses are so numerous as to display an intimate relationship between the sinus and all of the nerves and blood vessels in the sphenoid area. The internal carotid artery leads the others not only in frequency but also in degree of projection into the sinus. A carotid elevation was present in 65 of the 100 specimens. In 53, the condition was pronounced. In 14 of the 53, the vessel could be traced throughout its entire serpentine course along the sinus wall.” Sjöqvist\(^2\) reported a case in which angiography was performed after iodized oil

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\(^1\) Van Alyea

\(^2\) Sjöqvist
(Lipiodal) had been introduced into the sphenoidal sinus. Only a thin layer of bone was seen to intervene between the aneurysm and the sinus. A case of aneurysm in the sphenoidal sinus was described by Hirsch. A tumor was identified by x-ray within the sphenoidal sinus of a 53-year-old woman; there was no history of trauma to the head or epistaxis. This was incised via a transnasal approach, resulting in a fatal hemorrhage.

**Clinical Signs and Symptoms**

With increased awareness that an aneurysm can be produced from an injury to the head, many minor or insignificant clinical symptoms demand the attention of the physician. The fully ruptured aneurysm announces itself with catastrophic suddenness. The patient abruptly complains of a splitting headache, followed by vomiting, syncope, meningismus, confusion, coma, and death.

The more discrete initial and subsequent clinical symptoms of the traumatic aneurysms of the internal carotid artery usually depend on its localization.

According to Jaeger, aneurysm in the carotid canal causes symptoms only when the lesion is of large size. Sharp head pains and headache about the forehead and eyeball are common. This suggests that paralysis of the fourth or sixth cranial nerve with a possible concomitant Horner's syndrome may be the result of an aneurysm in the carotid canal. If the aneurysm develops anteriorly near or at the foramen lacerum, it lies close to the Gasserian ganglion and its branches. Consequently, corneal hypepthalgesia, anesthesia, or hyperesthesia of the infraorbital nerves, or even complete paralysis of the trigeminal nerve can occur. Pain may be projected into the orbit, inner canthus, forehead, and along the side of the nose. Bonnet mentioned the point that when part of the trigeminal symptomatology is associated with paralysis of one or more of the eye muscles, aneurysm of the carotid artery at or near the foramen lacerum is a good possibility. When the association is with a seventh or eighth nerve paralysis, one must think of an acoustic neurinoma. When the aneurysm occurs more distally, involvement of the oculomotor nerve is almost a constant feature. This is demonstrated by complete or partial ptosis of the upper lid so as to obscure vision; the eye turns outward; the pupil is dilated and does not react to light, convergence, or consensually. Jaeger stated that this paralysis may be periodic, usually lasting many weeks, then leaving for a period of weeks or months to recur. The fourth or sixth cranial nerves can be involved in this area as well. Unilateral exophthalmos may occur, along with cephalic bruit and possible pulsations. As the aneurysm is found more superiorly and anteriorly, pressure on the optic nerve or the optic chiasm can result in sudden or gradual unilateral visual failure and exophthalmos.

Meadows emphasized the principal clinical symptoms in relation to the direction of expansion of an aneurysm in the cavernous sinus. He stated that "with gradual enlargement, there is severe unilateral trigeminal pain, followed by involvement of one or more of the third, fourth and sixth cranial nerves. The pain may be limited to the distribution of the first division of the fifth nerve and a corresponding cutaneous sensory loss is common with impairment or loss of the corneal reflex. Posterior expansion of the aneurysm involves the second or third division of the fifth nerve. Anterior expansion leads to pressure on the optic nerve with gradual unilateral visual failure and exophthalmos. Medial expansion can result in pressure on the optic chiasm or nerve, with bilateral hemianopsia or unilateral visual failure with perhaps severe pain."

In an analysis of 31 cases of intracranial carotid aneurysm, Jaeger found that the combined symptoms of oculomotor paralysis or paresis associated with frontal or orbital head pains are sufficient to permit a presumptive diagnosis.
Brihaye et al. have established the following criteria for trauma as the cause of an intracranial aneurysm:

1. Trauma must affect the head.
2. Symptoms of concussion must exist after the trauma.
3. Between trauma and demonstration of the aneurysm, bridge symptoms like headache, epileptic attacks, fatigue, paralysis, and others must exist. (We should like to add the possibility of epistaxis as well.)
4. The cardiovascular system must be normal.
5. The presence of multiple aneurysms excludes a traumatic etiology.
6. The rupture of the aneurysm must be separated by a latent period of at least several days.
7. Rupture must occur without a new trauma.
8. The presence of hemosiderin pigment at the level of the aneurysm at autopsy suggests previous trauma.

With these criteria in mind, 10 cases were found, including our own, in which epistaxis was the result of a traumatic aneurysm (Table).

### Report of Case

A 29-year-old white man was rendered unconscious in an automobile accident on Jan. 22, 1957, for a short undetermined period of time.

Physical examination, soon after the accident, revealed a laceration across the left eyebrow extending to the periosteum of the skull. The pupil on the right reacted to light and accommodation; the pupil on the left was dilated and fixed, the eye pointing upward and laterally. There was bleeding from the nasal passages. The patient apparently had had an active epistaxis before entering the emergency room, but there was no leakage of cerebrospinal fluid. Examination revealed only mental confusion. Deep tendon reflexes were equal and physiologic. Roentgenograms showed nondisplaced fractures of the fourth, fifth, and sixth ribs on the right in the anterior axillary line. There were multiple fine-branching fractures of the left frontal bone extending into the roof of the left orbit and ending in the superoposterior portion of the left orbital cavity, the most superior of these fracture lines extending into the superior portion of the left frontal sinus. The right knee revealed cortical infarctions and tiny chip fractures in the medial tibial plateau.

One month after the accident the patient was transferred to this hospital. He was weak, complained of a poor appetite, frontal headaches, and paresthesia of the left frontal region. He had been having minor nosebleeds since the accident almost daily. The patient was known to have had grand mal epilepsy since the age of 11 years, but had...
Physical examination revealed a blood pressure of 120/80 mm. Hg. The patient was pale, oriented, and cooperative. Fresh blood clots were present in the left posterior nasopharynx. The left eye was totally ptotic. A scar extended from the medial aspect of the left brow, to the left lateral canthus. The left eye showed normal fourth and sixth cranial nerve function. It could not be elevated or depressed to the left nor could it be brought past the midline nasally. There was no nystagmus. The right pupil reacted to light and accommodation but not consensually. The left pupil was larger than the right. The results of visual acuity tests without correction were: O. D., 20/15; O. S., no light perception, not improvable. The left fundus showed a disc which was faintly paler than the right disc, but which was not definitely atrophic. Vessels in the left fundus were slightly less prominent than those on the right. The left macula was red with a surrounding paler area, which in turn was surrounded by an area of scleral sclerosis extending from the intranasal quadrant.

An electroencephalogram showed an abnormal record with a slow wave defct in the left hemisphere. This may have been due to recent trauma, since the general pattern showed improvement compared to a tracing taken eleven years previously.

**Hospital Course.**—Nerve regeneration became apparent with slight elevation of the left eyelid and increase in ability to bring the eye medially. It was felt that no ophthalmic surgery was indicated until the patient had been observed for approximately one year. The symptoms of headache and anorexia rapidly improved. Vital signs remained stable during the hospital stay. Some blood was seen in the nasopharynx and oropharynx daily, but there was no evidence of any active bleeding point. No further treatment was necessary. The patient was discharged on March 8, 1957. Approximately two months after this discharge, he returned to the Clinic with a complaint of daily recurrent epistaxis from the left naris, increasing in frequency for approximately two weeks. He was readmitted to the hospital on June 12, 1957.

On admission the patient stated that three days ago he had had a massive nasal hemorrhage which necessitated admission to another hospital. His blood pressure was then 75/50 mm. Hg. Five hundred cubic centimeters of whole blood and 500 cubic centimeters of dextran (Plavolex) were administered immediately, and the blood pressure rose to 100/60 mm. Hg. He did not bleed while in that hospital. The red blood count was 3,500,000 per cubic millimeter; hemoglobin 9.3 gm., white blood count 11,400 per cubic millimeter.

The physical examination revealed blood pressure of 100/66 mm. Hg. The left pupil was dilated and fixed. There was no vision in the left eye, and there was ptosis of the left upper lid. The nose revealed a deviated septum to the left with a sharp vomerian ridge. No bleeding point would be visualized. Dried blood was noted in both choanae.

Soft tissue studies of the neck and Waters projection of the paranasal sinuses revealed no evidence of encroachment on the air column and nasopharynx. No abnormalities were visualized in the neck. The paranasal sinuses were normal. Hematocrit value on June 18, 1957, was 31%; hemoglobin 11.8 gm. per 100 cc. of blood.

He continued to have mild epistaxis and occasional throbbing headaches on the left side, relieved on several occasions by meperidine (Demerol) intramuscularly. Because of the patient's poor general condition and anemia, the contemplated submucous resection was deferred. He bled almost daily for a brief period of time from the left nostril, requiring occasional packing with petrolatum gauze. A repeat electroencephalogram revealed a slow wave defect on the left side about the same as that seen in tracings done on March 1, 1957.

During his hospital stay, he was out of bed, ambulatory, friendly and cooperative. On the eve-
ning of June 20, 1957, he again complained of throbbing headache and epistaxis from the nose. He then began to bleed so profusely from his mouth and nose that suction could not evacuate the blood. The patient became exsanguinated.

Postmortem Examination.—There were found an obliterative fibrous pleuritis on the right, microscopic foci of fibrocaseous pulmonary tuberculosis, and pulmonary hemosiderosis.

The most significant changes were found in the cranial cavity. The left half of the anterior wall of the sella turcica and the greater portion of the roof of the sphenoid sinus were absent. A linear fracture was noted in the anterior fossa on the left, at the junction of the medial and lateral halves, extending anteriorly from the posterior margin for a distance of 1.25 cm. A hemorrhage was present in the wall of the sphenoid sinus.

The brain showed multiple areas of hemosiderin deposition superficially in the meninges of the left frontal lobe and left temporal lobe, with contusions in the underlying cerebrum.

There was a large aneurysm, roughly pyriform in shape, measuring 2.5 cm. in its greatest dimension from side to side, which arose from the left internal carotid artery. The aneurysm had extended medially and superiorly to lie medial and dorsal to the left optic nerve. The tuberculum

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Fig. 6.—Photomicrograph of internal carotid artery and aneurysm. A, mucous membrane of sphenoid sinus. B, wall of aneurysm. C, thrombus. D, bone. Reduced slightly from mag. X 100.
sellae and clinoid processes were eroded. The aneurysm had ruptured anteriorly and on its right lateral and ventral surfaces, where a large defect measuring 1 cm in diameter was present. This defect lay in relation to the sphenoid sinus cavity (Fig. 5).

Microscopic examination of the tissue at the site of the ruptured aneurysm disclosed a portion of the carotid artery in which the aneurysmal wall is thinned. The intima is slightly thickened by organized thrombus to which is adherent a large mass of thrombus material of varying age. The media, as well as the internal elastic lamina, has been destroyed and replaced by laminated layers of collagenous connective tissue. The latter lies in close apposition to the submucosal connective tissue of the sphenoid sinus, with no interposition of bone (Fig. 6).

Comment

Few traumatic aneurysms of the internal carotid artery have been reported in the literature, and rarely has epistaxis been noted as a conspicuous part of the clinical picture. Ten cases of epistaxis due to an intracranial aneurysm have been reported. Seven of these gave some history of previous trauma to the head. Considering the anatomy and mechanism of injury described below, epistaxis secondary to intracranial aneurysm must occur relatively frequently, and either has not been reported or has not been recognized. It is interesting to note that in 1928, in reporting a case of epistaxis due to a ruptured intracranial aneurysm, Birley 8 mentioned the relationship of the sphenoidal sinus to the middle fossa and stated: "It is therefore all the more curious that no example of epistaxis occurring as a late result of intracranial aneurysm, either traumatic or nontraumatic, appears to have been recorded."

DeVeer and Browder 9 maintained that in addition to rupture with immediate hemorrhage, or vascular occlusion by thrombosis, a third possible vascular lesion can occur; namely, aneurysmal dilatation of the injured vessel wall with rupture occurring days, weeks, or even months after injury. Hamby 10 stated that there is little doubt that trauma can cause aneurysms of the internal carotid artery, or that the artery itself can rupture into the cavernous sinus, producing an arteriovenous communication. However, he added that aneurysms of the intracranial vessels that are caused by trauma are extremely rare. In a more recent paper, Brihaye, Mage, and Verriest 7 mentioned that the hypothesis of torsion of the vessel followed by tearing of its wall, as a pathogenic mechanism of traumatic aneurysm, is strengthened by studies of the cerebral alterations in electrocuted criminals. Nonspecific parenchymatous alterations similar to those seen after head trauma were encountered. Tears of the vascular walls were frequent, generally of the large basal vessels, usually involving the elastic layer, but also the muscular layer and even the adventitia. This was attributed to the fact that the electric current travels along the vessels and provokes their contraction, torsion, and separation from the surrounding pia.

There are a number of factors that can probably influence occurrences of intermittent bleeding, such as fluctuation of the systolic and diastolic pressure, the oxygen and carbon dioxide content of the blood and surrounding tissues, hormonal influences as governed by the patient's emotions, atmospheric conditions, and perhaps certain idiopathic conditions that have not been elucidated. However, Richardson and Hyland 11 found that the activity of the patient at the time of hemorrhage was of little importance in determining its onset or severity. The patients were at rest in bed, standing, or walking in 78% of the cases; undue muscular exertion appeared to precipitate the onset in only about 18%. The authors concluded that the rupture of an aneurysmal sac is usually a gradual process of stretching and hemorrhagic dissection, and that in only a few cases is a tear completed by sudden increase in blood pressure due to violent muscular effort. In Hamby's 10 series most of the patients were at their usual work, driving a car, walking, sitting, standing, and even in several instances, asleep. Hamby also felt that the patients' activity at the time of onset of bleeding was unimportant.
RUPTURED INTRACRANIAL ANEURYSM

Epistaxis may occur as a result of the erosion and rupture of an aneurysm into the sphenoid sinus, the Eustachian tube, or the cribiform plate.

It is well known that aneurysms or abnormal blood vessels can gradually erode through solid bone. This is readily seen where an aortic aneurysm has eroded the bodies of vertebrae. In coarctation of the aorta notchering or scalloping under the lower edges of the ribs results from the increased size, tortuosity, and elongation of the intercostal arteries.

Aneurysms, traumatic or otherwise, tend to occur in the cavernous portion or superior extracavernous portion of the internal carotid artery. Epistaxis by erosion into the sphenoid sinus is the one most frequently encountered. Of incidental interest is a statement by Davis that the sphenoid air sinus is opened in about 70% of fractures of the middle fossa of the skull. Figures 3 and 4 demonstrate that the artery lies in close approximation to the lateral wall of the sphenoid sinus and may even groove the wall. An aneurysm in this location or one that is extended to this area from several millimeters distant can easily erode the thin lateral wall of the sinus. In many skulls which we personally examined, the bony wall was less than 2 mm. in thickness. The process of bleeding and clotting within the sinus can readily produce intermittent epistaxis via the sphenoid ostia.

The second possible site of nasal bleeding is the traumatic aneurysm of the carotid artery within the carotid canal. Here again, there is only a thin lamella of bone between the carotid canal and the Eustachian tube (Fig. 2). There may be only a thin, bony cribiform type of barrier between them. A rupture here will produce posterior pharyngeal bleeding and anterior epistaxis. Rousseau and Spillman reported such a case where nasopharyngoscopy revealed some blood clots around and in the Eustachian tube orifice.

The third possibility, which has never been reported and is the least likely to occur, but is quite feasible anatomically, is bleeding through the cribiform plate. A lesion of one of the anterior cerebral vessels or its branches with an associated tear in the dura will produce epistaxis via the cribiform plate. A fracture line through the lesser wing of the sphenoid, in proximity to an aneurysm of the cavernous or extracavernous portion of the internal carotid artery, extending into the orbit along the ophthalmic artery, can produce epistaxis via the ethmoidal labyrinth or cribiform plate.

Summary and Conclusions

Traumatic aneurysm of the internal carotid artery is primarily a neurosurgical problem. The initial clinical symptoms have been reviewed. Such aneurysms are not common, and even more unusual is epistaxis as a major symptom. Nine reported cases in the literature have been cited, of which one was confirmed by autopsy.

We have demonstrated how trauma to the head can produce an intracranial aneurysm, which in turn can lead to epistaxis. The epistaxis can originate in three possible sites: the sphenoid sinus, the Eustachian tube, or the cribiform plate. The otolaryngologist may be the first to examine such a patient. It is important to recognize that epistaxis may be the clue to the diagnosis of ruptured intracranial aneurysm.

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REFERENCES


Seftel et al.


